Complete Summary

GUIDELINE TITLE

Inpatient management of heart failure.

BIBLIOGRAPHIC SOURCE(S)

Institute for Clinical Systems Improvement (ICSI). Inpatient management of heart failure. Bloomington (MN): Institute for Clinical Systems Improvement (ICSI); 2004 Feb. 85 p. [169 references]

COMPLETE SUMMARY CONTENT

SCOPE

DISEASE/CONDITION(S)

Heart failure including acute pulmonary edema

GUIDELINE CATEGORY

Diagnosis
Evaluation
Management
Treatment

CLINICAL SPECIALTY

Cardiology
Emergency Medicine
Family Practice
Internal Medicine

INTENDED USERS

Advanced Practice Nurses
Allied Health Personnel
Health Care Providers
Health Plans
Hospitals
Nurses
Physician Assistants
Physicians

GUIDELINE OBJECTIVE(S)

- To reduce the mortality and morbidity from heart failure by improving the pharmacologic treatment of patients with heart failure
- To improve the treatment of patients with heart failure by assuring that patients have the etiology and/or precipitating factors of heart failure identified
- To improve care of patients with heart failure by assuring comprehensive follow-up care
- To improve care of patients with heart failure by integrating patient feedback
- To improve care of patients with heart failure by decreasing the number of hospitalizations of patients with heart failure

TARGET POPULATION

Adult patients with heart failure requiring hospitalization

INTERVENTIONS AND PRACTICES CONSIDERED

Inpatient Management Algorithm

1. Inpatient assessment, including history and physical examination, laboratory and diagnostic tests (including measurement of brain natriuretic peptide,) assessment of causative and precipitating factors, corrections of systemic factors
2. Pharmacologic treatment and volume management including:
    - Vasodilators -- angiotensin-converting enzyme [ACE] inhibitors, such as captopril [Capoten], enalapril [Vasotec], enalaprilat [Vasotec IV], lisinopril [Prinivil, Zestril], benazepril [Lotensin], fosinopril [Monopril], quinapril [Accupril], moexipril [Univasc], trandolapril [Mavik]. ramipril [Altace]; angiotensin II receptors blockers, such as candesartan [Atacand], eprosartan [Teveten], irbesartan [Avapro], losartan [Cozaar], olmesartan [Benicar], telmisartan [Micardis], valsartan [Diovan]; hydralazine/isosorbide dinitrate; nitroglycerin; nitroprusside; nesiritide [Natrecor]
    - Diuretics -- thiazide diuretics; loop diuretics, such as furosemide, bumetanide, ethacrynic acid, and torsemide; thiazide-related diuretics, such as metolazone; potassium-sparing diuretics, such as spironolactone, triamterene, amiloride, and eplerenone
    - Inotropes -- digoxin, dobutamine [Dobutrex], dopamine [Intropin], milrinone [Primacor]
    - Beta blockers -- metoprolol tartrate, metoprolol succinate (Toprol XL), carvedilol (Coreg), bisoprolol fumarate (Zebeta), atenolol

- Spironolactone
- Antiarrhythmics
- Anticoagulants (warfarin)

3. Non-invasive and invasive imaging/physiologic assessment, including electrocardiogram, chest x-ray, and assessment of ventricular function (echocardiogram, radionuclide ventriculography)
4. Non-pharmacologic management, including patient education, diet and lifestyle counseling, exercise program, stress reduction

Emergent Management Algorithm

1. Initial patient assessment, including history, physical exam, and differential diagnosis
2. Initiation of O_2 therapy, intravenous fluids, labs, chest x-ray, electrocardiogram , echocardiogram
3. Assessment of blood pressure, perfusion, and volume status
4. Vasodilators, inotropes, specialty consultation, mechanical assist devices

Acute Pulmonary Edema Algorithm

1. Loop diuretics (furosemide [Lasix], bumetanide [Bumex], torsemide [Demadex])
2. Nitroglycerine sublingual or paste
3. Nitroglycerine infusion
4. Nesiritide
5. Intensive care unit admission, specialty consultation, tertiary center referral

MAJOR OUTCOMES CONSIDERED

- Sensitivity and specificity of clinical assessments, laboratory evaluations, and other diagnostic tests
- Effectiveness and safety of pharmacologic treatment and volume management in patients with heart failure
- All-cause mortality, cardiovascular mortality, and hospitalization rates in heart failure patients

METHODOLOGY

METHODS USED TO COLLECT/SELECT EVIDENCE

Searches of Electronic Databases

DESCRIPTION OF METHODS USED TO COLLECT/SELECT THE EVIDENCE

Not stated

NUMBER OF SOURCE DOCUMENTS

Not stated

METHODS USED TO ASSESS THE QUALITY AND STRENGTH OF THE EVIDENCE

Weighting According to a Rating Scheme (Scheme Given)

RATING SCHEME FOR THE STRENGTH OF THE EVIDENCE

Key conclusions (as determined by the work group) are supported by a conclusion grading worksheet that summarizes the important studies pertaining to the conclusion. Individual studies are classed according to the system presented below, and are designated as positive, negative, or neutral to reflect the study quality.

Conclusion Grades:

Grade I: The evidence consists of results from studies of strong design for answering the question addressed. The results are both clinically important and consistent with minor exceptions at most. The results are free of any significant doubts about generalizability, bias, and flaws in research design. Studies with negative results have sufficiently large samples to have adequate statistical power.

Grade II: The evidence consists of results from studies of strong design for answering the question addressed, but there is some uncertainty attached to the conclusion because of inconsistencies among the results from the studies or because of minor doubts about generalizability, bias, research design flaws, or adequacy of sample size. Alternatively, the evidence consists solely of results from weaker designs for the question addressed, but the results have been confirmed in separate studies and are consistent with minor exceptions at most.

Grade III: The evidence consists of results from studies of strong design for answering the question addressed, but there is substantial uncertainty attached to the conclusion because of inconsistencies among the results of different studies or because of serious doubts about generalizability, bias, design flaws, or adequacy of sample size. Alternatively, the evidence consists solely of results from a limited number of studies of weak design for answering the question addressed.

Grade Not Assignable: There is no evidence available that directly supports or refutes the conclusion.

Study Quality Designations:

The quality of the primary research reports and systematic reviews are designated in the following ways on the conclusion grading worksheets:

Positive: indicates that the report or review has clearly addressed issues of inclusion/exclusion, bias, generalizability, and data collection and analysis.

Negative: indicates that these issues (inclusion/exclusion, bias, generalizability, and data collection and analysis) have not been adequately addressed.

Neutral: indicates that the report or review is neither exceptionally strong nor exceptionally weak.

Not Applicable: indicates that the report is not a primary reference or a systematic review and therefore the quality has not been assessed.

Classes of Research Reports:

A. Primary Reports of New Data Collection:

Class A:

- Randomized, controlled trial

Class B:

- Cohort study

Class C:

- Non-randomized trial with concurrent or historical controls
- Case-control study
- Study of sensitivity and specificity of a diagnostic test
- Population-based descriptive study

Class D:

- Cross-sectional study
- Case series
- Case report

B. Reports that Synthesize or Reflect upon Collections of Primary Reports:

Class M:

- Meta-analysis
- Systematic review
- Decision analysis
- Cost-effectiveness analysis

Class R:

- Consensus statement
- Consensus report
- Narrative review

Class X:

- Medical opinion

METHODS USED TO ANALYZE THE EVIDENCE

Review of Published Meta-Analyses
Systematic Review with Evidence Tables

DESCRIPTION OF THE METHODS USED TO ANALYZE THE EVIDENCE

Not stated

METHODS USED TO FORMULATE THE RECOMMENDATIONS

Not stated

RATING SCHEME FOR THE STRENGTH OF THE RECOMMENDATIONS

Not applicable

COST ANALYSIS

A formal cost analysis was not performed and published cost analyses were not reviewed.

METHOD OF GUIDELINE VALIDATION

Clinical Validation-Pilot Testing
Internal Peer Review

DESCRIPTION OF METHOD OF GUIDELINE VALIDATION

Institute Partners: System-Wide Review

The guideline draft, discussion, and measurement specification documents undergo thorough review. Written comments are solicited from clinical, measurement, and management experts from within the member medical groups during an eight-week period of "Critical Review."

Each of the Institute's participating medical groups determines its own process for distributing the guideline and obtaining feedback. Clinicians are asked to suggest modifications based on their understanding of the clinical literature coupled with their clinical expertise. Representatives from all departments involved in implementation and measurement review the guideline to determine its operational impact. Measurement specifications for selected measures are developed by the Institute for Clinical Systems Improvement (ICSI) in collaboration with participating medical groups following general implementation of the guideline. The specifications suggest approaches to operationalizing the measure.

Guideline Work Group: Second Draft

Following the completion of the "Critical Review" period, the guideline work group meets 1 to 2 times to review the input received. The original guideline is revised as necessary, and a written response is prepared to address each of the

suggestions received from medical groups. Two members of the Cardiovascular Steering Committee carefully review the Critical Review input, the work group responses, and the revised draft of the guideline. They report to the entire committee their assessment of two questions: (1) Have the concerns of the medical groups been adequately addressed? (2) Are the medical groups willing and able to implement the guideline? The committee then either approves the guideline for pilot testing as submitted or negotiates changes with the work group representative present at the meeting.

Pilot Test

Medical groups introduce the guideline at pilot sites, providing training to the clinical staff and incorporating it into the organization's scheduling, computer, and other practice systems. Evaluation and assessment occur throughout the pilot test phase, which usually lasts for three months. Comments and suggestions are solicited in the same manner as used during the "Critical Review" phase.

The guideline work group meets to review the pilot sites' experiences and makes the necessary revisions to the guideline, and the Cardiovascular Steering Committee reviews the revised guideline and approves it for implementation.

RECOMMENDATIONS

MAJOR RECOMMENDATIONS

The recommendations for inpatient management of heart failure are presented in the form of an algorithm with 48 components, accompanied by detailed annotations. Algorithms are provided for: Inpatient Management, Emergent Management, and Acute Pulmonary Edema; clinical highlights and selected annotations (numbered to correspond with the algorithm) follow.

Class of evidence (A-D, M, R, X) ratings and key conclusion grades (I-III, Not Assignable) are defined at the end of the "Major Recommendations" field.

Clinical Highlights and Recommendations

1. Evaluate patients presenting with heart failure for evidence of secondary ischemic heart disease and exacerbating and underlying causes of heart failure. (Annotation #4)
2. After evaluation, diagnosis, and initiation of pharmacological management of heart failure, follow-up in the ambulatory setting focuses on optimizing pharmacological therapy and preventing heart failure exacerbations. Patient education is central in this effort. The core of patient education is dietary and lifestyle management including monitoring daily weights, fluid management, sodium restriction, recognition of symptoms and early intervention, adherence with the treatment plan, modification of dietary and alcohol intake, exercise, and stress reduction. (Annotation #9)
3. Treat all patients, including Class IV patients, with left ventricular systolic dysfunction with angiotensin-converting enzyme (ACE) inhibitors unless specific contraindications exist. (Annotation #5)

4. Treat all patients, including Class IV patients, with beta blockers unless specific contraindications exist. (Annotation #5)
5. Consider early specialty referral for patients with ischemia or those who are refractory despite optimal medical therapy. (Annotation #8)
6. Brain natriuretic peptide (BNP) is useful in the diagnosis of heart failure in patients with dyspnea of unknown etiology. Intravenous Nesiritide (Natrecor®) is effective and may be used to treat patients with acutely decompensated heart failure. (Annotations #4, 5)

Inpatient Management Algorithm Annotations

1. Signs and Symptoms of Heart Failure (Excluding Acute Coronary Syndrome)

   Refer to the original guideline document for detailed information on the signs and symptoms of heart failure.

2. Hospital Management Required?

   Consider hospitalization in the presence or suspicion of heart failure with any of the following findings:

   - Clinical, laboratory, or electrocardiographic evidence of acute myocardial ischemia or infarction
   - Pulmonary edema or severe respiratory distress
   - Severe complicating medical illness (e.g., pneumonia, renal failure)
   - Anasarca (generalized edema)
   - Symptomatic hypotension or syncope
   - Symptoms refractory to outpatient therapy
   - Thromboembolic complications requiring interventions
   - Management of clinically significant arrhythmias (hemodynamic effects)
   - Inadequate social support for safe outpatient management
   - Hyperkalemia

4. Inpatient Assessment

   By definition, these patients are Stage C and D, New York Heart Association (NYHA) Class III or IV. Heart failure should not be the final, stand-alone diagnosis. There should always be an associated etiology and/or contributing factor. The etiology of heart failure and the presence of exacerbating factors or other diseases that may have an important influence on management should be carefully considered in all cases. Refer to Annotation Appendix B, "Heart Failure Classification" in the original guideline document.

   A. History:
      - Previous diagnosis
      - Blunt chest injury
      - Confusion
      - Causes of low hemoglobin (if anemic)
      - Recent weight gain

- Symptoms of thyroid dysfunction
- Degree of exercise limitation (exertional dyspnea)
- Alcohol use
- Positive cardiac risk factors (smoking, diabetes, hyperlipidemia, family history, male gender, family history of heart failure or congenital heart disease)
- Other medical conditions (recent pregnancy)
- History of hypertension
- Screen for depression
- Angina/chest pain/previous history of coronary artery disease/peripheral vascular disease
- Assess for venous thromboembolism
- Palpitations
- Recent viral infection
- Rheumatic fever
- History of human immunodeficiency virus (HIV)
- Bacterial endocarditis
- Evaluation of medication/treatment plan adherence (recent medication changes, over-the-counter medication)
- Shortness of breath
- Foreign travel

B. Physical Exam:
    - Vital signs including weight and height
    - Abdomen: large, pulsatile, or tender liver or ascites
    - Diaphoresis
    - Lower extremity edema in the absence of venous insufficiency
    - Elevated jugular venous pressure, positive hepatojugular reflux
    - Diminished peripheral pulse
    - Heart sounds: S3, S4, or murmur
    - Lungs: labored breathing, rales above the lower 25% of the lung that do not clear with cough
    - Left lateral displacement of point of maximal impulse (PMI)
    - Skin color: cyanosis, pallor, jaundice
    - Heart rate: Tachycardia, bradycardia/arrhythmias
C. Laboratory Evaluation: (Those not performed in the emergency room [ER])
    - Complete blood count (CBC)
    - Electrolytes (add MG^{++} if on diuretics or have ventricular arrhythmias)
    - Iron/iron binding capacity (IBC) (if suspect cardiomyopathy)
    - Urinalysis
    - Liver function
    - Tests for myocardial injury: Troponin, creatine kinase/creatine kinase MB band (CK/CKMB)
    - Renal function
    - Screening thyroid-stimulating hormone (TSH) (if new onset of heart failure)
    - Lipid profile
    - Arterial blood gases
    - Brain natriuretic peptide (BNP) (for differential diagnosis)
    - Blood cultures (if suspect bacterial endocarditis)
    - Lymes serology (if suspect bradycardia/heart block)

Brain natriuretic peptide (BNP) has been found useful in the diagnosis of patients with dyspnea of unknown etiology. The BNP test is helpful in ruling out a cardiac cause when the BNP level is less than 100. BNP is correlated with severity of heart failure in patients with heart disease. There is currently insufficient evidence to support the use of BNP to monitor patient therapy. [Conclusion Grade I: See Conclusion Grading Worksheet – Appendix A – Annotation #4 (BNP) in the original guideline document].

Evidence supporting this recommendation is of classes: A, C, R

When suspected, screening for uncommon causes such as hemochromatosis, HIV, connective tissue disease, and pheochromocytoma should be carried out.

D. Diagnostic Tests:
- Electrocardiogram (ECG)
- Assessment of ventricular function (echocardiogram, radionuclide ventriculography)
- Chest radiograph
- Ischemia evaluation in patients with coronary artery disease (CAD) risk factors (stress test, angiography)
- Rhythm monitoring
- Myocardial biopsy

An electrocardiogram and chest radiograph are fundamental parts of the initial evaluation for heart failure. In addition, the objective evaluation of ventricular performance is also a critical part for patients with suspected or known heart failure. Objective assessment of left ventricular (LV) function is necessary because chest x-ray (CXR), ECG, and history and physical (H&P) often fail to distinguish normal from low ejection fraction (EF) in patients with heart failure.

Magnetic resonance imaging or computed tomography may be useful in evaluating ventricular mass, detecting right ventricular dysplasia, or recognizing the presence of pericardial disease.

In patients with CAD and angina, patients with suspected CAD as a cause of heart failure, or patients with LV dysfunction but without angina, coronary angiography may be the investigation of choice to determine coronary anatomy and the need for revascularization. In patients in whom coronary artery disease has been excluded previously as the cause of left ventricular dysfunction, repeated invasive or noninvasive assessment for ischemia is generally not indicated.

Evidence supporting this recommendation is of classes: D, M

Refer to the Institute for Clinical Systems Improvement (ICSI) guideline Heart Failure in Adults: Annotation #3, Conclusion Grade II.

E. Assess for causative and precipitating factors

Causes of heart failure can be classified as cardiac and non-cardiac. Refer to tables at end of Annotation #4 in the original guideline document for the salient features of the more common causes.

It is important to make a determination whether heart failure is due to systolic dysfunction or diastolic dysfunction. One-third of patients have predominantly diastolic dysfunction, one-third have both diastolic and systolic dysfunction, and one-third have predominantly systolic dysfunction.

Ischemia is responsible for the majority of cases of heart failure. Two-thirds of systolic heart failure is due to ischemic heart disease. Identifying ischemia as a cause of heart failure is important, as a majority of these patients would benefit from revascularization.

The distinction between systolic and diastolic dysfunction is important because the choice of therapy may be quite different and some therapies for systolic dysfunction may even be harmful if used to treat diastolic dysfunction.

Patients present with heart failure in one of the following ways:

1. Decreased effort tolerance – dyspnea, fatigue
2. Volume overload
3. Presentation as comorbidity with other cardiac or non-cardiac conditions. (AHA)

Of these presentations, the syndrome of volume overload with pulmonary edema presents the most urgent scenario in the inpatient setting. Cardiogenic pulmonary edema may be a manifestation of either left atrial outflow impairment or left ventricular systolic or diastolic dysfunction.

Left atrial outflow impairment – In chronic left atrial outflow impairment, pulmonary edema is often precipitated when an elevated heart rate (e.g., associated with atrial fibrillation, ventricular tachycardia, exertion, fever, stress) decreases the time for left ventricular filling. It may also be precipitated by an increased vascular volume, as occurs with pregnancy or an increase in salt intake. Mitral stenosis secondary to rheumatic heart disease is a common but decreasing cause of impaired left atrial outflow. Other causes include left atrial tumors (most commonly a myxoma), a thrombus of a prosthetic valve, and a congenital membrane in the left atrium (e.g., cor triatriatum).

Pulmonary edema may also result from acute left atrial outflow impairment due to acute mitral valve insufficiency. Acute mitral valve insufficiency can result from papillary muscle dysfunction or rupture of

the chordae tendineae, most often following an acute myocardial infarction.

Left ventricular dysfunction – Left ventricular systolic and diastolic dysfunction, left ventricular volume overload, and left ventricular outflow obstruction may all lead to pulmonary edema.

Left ventricular volume overload – Left ventricular volume overload can be induced by ventricular septal rupture, aortic insufficiency, arthrogenic fluid overload, and, as noted above, primary sodium retention due to renal disease. Acute ventricular septal rupture is a complication of acute myocardial infarction (both anterior and inferior).

With aortic insufficiency, either acute or chronic, the valvular dysfunction results in an acute increase in left ventricular volume, with resultant elevation of left ventricular end-diastolic and left atrial pressures and pulmonary edema. Acute aortic insufficiency is usually the result of infective endocarditis, acute aortic dissection, or trauma.

Left ventricular outflow obstruction – Left ventricular outflow obstruction can be the result of critical aortic stenosis (including supravalvular and subvalvular stenosis), hypertrophic cardiomyopathy, and/or severe systemic hypertension. Chronic left ventricular outflow obstruction is associated with a hypertrophied left ventricular wall, which can produce diastolic and/or systolic dysfunction.

Clinical Picture of Myocarditis – The usefulness of endomyocardial biopsy in the evaluation of patients with a cardiomyopathy of unknown cause is not clear. Biopsy findings frequently do not have a material effect on patient management. Many patients with suspected myocarditis improve with supportive care without anti-inflammatory or anti-viral treatment. Immunosuppressive therapy does not appear to influence prognosis of chronic cardiomyopathy.

Myocardial biopsy may be helpful in diagnosing hemochromatosis, sarcoidosis, endocardial fibroelastosis, amyloidosis, etc. When these conditions or when the need for myocardial biopsy is considered, refer to a specialist.

F. Correction of Systemic Factors

Treatment is driven by the particular disease or clinical state. Specific treatment modalities for secondary causes of heart failure are considered outside the scope of this guideline.

A search for reversible exacerbating factors is important. Arrhythmias or ischemia may cause heart failure, pulmonary edema, or shock with or without any major cardiac dysfunction. Myocardial ischemia, changes in valvular regurgitation, pulmonary embolism, infection, renal dysfunction, side effects of drugs, and excessive fluid, sodium,

and alcohol may cause or exacerbate symptoms of heart failure in patients with existing cardiac dysfunction.

5. Pharmacologic Treatment and Volume Management

Angiotensin converting enzyme inhibitors, angiotensin receptor blockers, beta blockers, and spironolactone belong to a larger class of medications entitled neurohormonal antagonists.

Vasodilators

Angiotensin-Converting Enzyme (ACE) Inhibitors:

- ACE inhibitors should be prescribed for all patients with left ventricular systolic dysfunction unless specific contraindications exist. Contraindications include:
    1. History of intolerance or adverse reactions to these agents
    2. Serum potassium greater than 5.5 meq/L
    3. Symptomatic hypotension (unless due to excessive diuresis)
    4. Severe renal artery stenosis
    5. Pregnancy
    6. Cough and rash side effects
    7. Known hypersensitivity to ACE inhibitors
- To achieve the full mortality reductions possible with ACE inhibitors, the dose must be titrated to the moderate to high dose range (e.g., 20-40 mg Lisinopril once daily [QD]). Lower dose therapy has been shown to be less effective in reducing mortality.
- Approach to initiating ACE inhibitor therapy:

    1. Start at low dose and titrate upward over several weeks to achieve a decrease in blood pressure
    2. Consider holding one dose of diuretic before giving the first dose of ACE inhibitors, particularly in patients with low baseline blood pressure.
- Patients being actively titrated on ACE inhibitors will need to be seen frequently to monitor their blood pressure, potassium, and renal function.
- Hypotension: Patients should be well hydrated before initiation or increase of ACE inhibitors. If the patient develops hypotension in the absence of hypovolemia, splitting the dose or switching from morning (a.m.) to bedtime (h.s.) dosing (in long-acting agents) may be helpful. If this is ineffective, the dose should be reduced to the highest dose tolerated.
- Hyperkalemia: If potassium is high in the absence of supplementation, the ACE inhibitor should be discontinued for 3 days, then restarted at the last dose tolerated. Digoxin toxicity and renal insufficiency should also be considered.
- Renal Insufficiency: Blood urea nitrogen (BUN) and creatinine should be monitored regularly in patients on ACE inhibitors, and more frequently during active titration. An increase in serum creatinine of 0.5 mg/dL or more is an indication for reassessment of volume status. There is no absolute level of creatinine to preclude the use of ACE

inhibitors. Caution should be exercised if used in patients with elevated serum creatinine.
- All ACE inhibitors that have been studied to date in treatment of heart failure have shown benefit. Therefore, simpler dosing regimens may be equally effective and less expensive. ACE inhibitors slow disease progression, improve exercise capacity, and decrease hospitalization and mortality.
- See Annotation Appendix C, "Comparison of Approved ACE Inhibitors" in the original guideline document.

Refer to the NGC summary of the ICSI guideline Heart Failure in Adults Annotations #6, 14 (ACE Inhibitors).

Evidence supporting this recommendation is of class: A

Angiotensin Receptor Blockers (ARB):

- Beneficial to reduce afterload and improve cardiac output
- Angiotensin receptor blockers should be considered instead of ACE inhibitors primarily in patients who are intolerant of ACE inhibitors because of angioedema or intractable cough. The work group prefers the use of this medication over hydralazine/isosorbide dinitrate because of its ease of use. This could potentially increase patient compliance.
- Direct comparison with regards to mortality showed no difference between Losartan and Captopril.
- Direct comparison with regards to efficacy and safety showed no difference between Valsartan and Enalapril. Contraindications include:

    1. History of intolerance or adverse reactions to these agents
    2. Serum potassium greater than 5.5 meq/L.
    3. Symptomatic hypotension (unless due to excessive diuresis)
    4. Severe renal artery stenosis
    5. Pregnancy
- Angiotensin receptor blockers are as likely as ACE inhibitors to produce hypotension, worsening renal function, and hyperkalemia.
- There are conflicting data when adding an ARB to an ACE inhibitor or to ACE inhibitor and a beta blocker. (See Discussion and References #5, "Angiotensin II Receptor Blockers" in the original guideline document).
- Benefit in heart failure patients with preserved LV systolic function (LV ejection fraction of >40%)
- See Appendix D, "Comparison of Approved Angiotensin II Receptor Antagonists" in the original guideline document.

Evidence supporting this recommendation is of classes: A, M

Hydralazine/Isosorbide Dinitrate:

- Hydralazine/isosorbide dinitrate may be considered as a therapeutic option in those patients experiencing hypotension or renal insufficiency secondary to ACE inhibitor usage.

- If the potassium continues to rise after titration of the ACE inhibitor dose, it should be discontinued and a combination of hydralazine/isosorbide dinitrate should be tried.

Evidence supporting this recommendation is of class: A

Nitroglycerin:

- Food and Drug Administration (FDA) Indication: Intravenous nitroglycerin is indicated for the treatment of heart failure in patients with concomitant acute myocardial infarction.
- Non-FDA Indication: Nitroglycerin can be used to treat pulmonary edema.
- Intravenous nitroglycerin is the only dosage form approved in the U.S. for use in heart failure associated with acute myocardial infarction, although sublingual, transmucosal, and transdermal dosage forms have been used for both acute and chronic symptomatic control.
- Nitroglycerin is normally reserved for patients whose cardiac index is adequate but pulmonary wedge pressure is elevated (greater than 18 mm Hg). A combination of diuretics and nitroglycerin or nitrates is effective in lowering pulmonary capillary wedge pressure.
- Inotropes such as dobutamine or dopamine can be safely added to those patients on intravenous nitroglycerin who need inotropic support.
- Although an unlabelled use, oral and transdermal nitrates have been used to reduce cardiac work in chronic heart failure NYHA class III and IV.
- Nitroglycerin is contraindicated in individuals with a known hypersensitivity or idiosyncrasy reaction to nitroglycerin, other organic nitrates, or nitrites. (PI)
- Nitroglycerin is also contraindicated in patients with:

    1. Hypotension or uncorrected hypovolemia, as the use of nitroglycerin in such states could produce severe hypotension or shock
    2. Increased intracranial pressure (e.g., head trauma or cerebral hemorrhage)
    3. Inadequate cerebral circulation
    4. Constrictive pericarditis and pericardial tamponade
- Precautions with nitroglycerin use include:

    1. The use of any form of nitroglycerin during the early days of acute myocardial infarction requires particular attention to hemodynamic monitoring and clinical status to avoid the hazards of hypotension and tachycardia.
    2. Severe hypotension, particularly with upright posture, may occur even with small doses of nitroglycerin. The drug, therefore, should be used with caution in subjects who may have volume depletion from diuretic therapy or in patients who have low systolic blood pressure (e.g., below 90 mm Hg). Paradoxical bradycardia and increased angina pectoris may accompany nitroglycerin-induced hypotension. Nitrate therapy

may aggravate the angina caused by hypertrophic cardiomyopathy.
3. Tolerance to this drug and cross-tolerance to other nitrates and nitrites may occur.
4. Nitroglycerin injection should be used with caution in patients who have severe hepatic or renal disease
5. Excessive hypotension, especially for prolonged periods of time, must be avoided because of possible deleterious effects on the brain, heart, liver, and kidney from poor perfusion and the attendant risk of ischemia, thrombosis, and altered function of these organs. Paradoxical bradycardia and increased angina pectoris may accompany nitroglycerin-induced hypotension. Patients with normal or low pulmonary capillary wedge pressure are especially sensitive to the hypotensive effects of nitroglycerin injection. If pulmonary capillary wedge pressure is being monitored, it will be noted that a fall in wedge pressure precedes the onset of arterial hypotension, and the pulmonary capillary wedge pressure is thus a useful guide to safe titration of the drug.

Evidence supporting this recommendation is of classes: A, D, M, R

Nitroprusside (Intravenous):

- Nitroprusside can be used for the treatment of symptoms associated with advanced, decompensated heart failure.
- This group would recommend nitroprusside only in those patients with side-effects and/or resistance to nitroglycerin or the presence of specific contraindications.
- Contraindications to using nitroprusside include:

    1. Using nitroprusside in the treatment of compensatory hypertension, where the primary hemodynamic lesion is aortic coarctation or arteriovenous shunting
    2. Using nitroprusside to produce hypotension during surgery in patients with known inadequate cerebral circulation or in moribund patients (A.S.A. Class 5E) coming to emergency surgery
    3. Nitroprusside should be avoided in patients with congenital (Leber's) optic atrophy or with tobacco amblyopia (a rare form of amblyopia combining nutritional deficiency and tobacco sensitivity). These patient populations have unusually high cyanide/thiocyanate ratios.
- Precautions with nitroprusside use include:

    1. Like other vasodilators, nitroprusside can cause increases in intracranial pressure. In patients whose intracranial pressure is already elevated, nitroprusside should be used only with extreme caution.
    2. Extended use of nitroprusside, especially with concomitant renal dysfunction can produce cyanide toxicity. This is normally avoided by mixing sodium thiosulfate in nitroprusside infusions.

3. Use caution when administering nitroprusside to patients with hepatic insufficiency.

Evidence supporting this recommendation is of classes: A, D, R

Nesiritide (Natrecor®):

- Nesiritide is indicated for the intravenous treatment of patients with acutely decompensated heart failure who have dyspnea at rest or with minimal activity.
- The best candidates for nesiritide therapy are patients with decompensated heart failure who have clinical evidence of fluid overload and/or raised central venous pressure.
- It is the opinion of this group that nesiritide be reserved for those acutely decompensated heart failure patients who are:

    1. Volume overloaded despite aggressive diuresis
    2. Displaying tolerance and/or resistance to vasodilators or diuretics
    3. Demonstrating significant side-effects to other vasodilators that are intolerable
- In comparison with dobutamine, nesiritide causes significantly less heart rate variances, tachycardia, premature ventricular beats, and repetitive beats.
- Contraindications to the use of nesiritide include:

    1. Hypersensitivity to nesiritide or any of its components
    2. Hypotension with a systolic blood pressure less than 90 mm Hg
    3. Using nesiritide in patients with cardiogenic shock
- Precautions with nesiritide use include:

    1. Parenteral administration of protein pharmaceuticals or Escherichia coli-derived products should be attended by appropriate precautions in case of an allergic reaction.
    2. Nesiritide is not recommended for patients for whom vasodilating agents are not appropriate, such as patients with significant valvular stenosis, restrictive or obstructive cardiomyopathy, constrictive pericarditis, pericardial tamponade, or other conditions in which cardiac output is dependent upon venous return, or for patients suspected to have low cardiac filling pressures.
    3. In patients with severe heart failure whose renal function may depend on the activity of the renin-angiotensin-aldosterone system, treatment with nesiritide may be associated with azotemia. When nesiritide was initiated at doses higher than 0.01 micrograms/kg/min, there was an increased rate of elevated serum creatinine over baseline compared with standard therapies.
    4. Nesiritide may cause hypotension. The hypotension induced by nesiritide in clinical studies was longer in duration than that of nitroglycerin. In earlier trials, when nesiritide was initiated at doses higher than the 2 micrograms/kg bolus followed by a

0.01 micrograms/kg/min infusion, there were more hypotensive episodes and these episodes were of greater intensity and duration. They were also more often symptomatic and/or more likely to require medical intervention. Therefore, nesiritide should be administered only in settings where blood pressure can be monitored closely, and the dose of nesiritide should be reduced or the drug discontinued in patients who develop hypotension. The rate of symptomatic hypotension may be increased in patients with a blood pressure <100 mm Hg at baseline and should be used cautiously in these patients. The potential for hypotension may be increased by combining nesiritide with other drugs that may cause hypotension.

5. There is little experience with infusions of nesiritide for more than 48 hours.

Evidence supporting this recommendation is of classes: A, R

Diuretics

- Patients with signs of volume overload should be started on a diuretic; however, this should not be the sole therapy
- Two recent studies have suggested that torsemide is better than furosemide in treating patients with heart failure.
- Severe volume overload, severe renal insufficiency (creatinine clearance less than 30 ml/min), or persistent edema despite thiazide diuretics are all indications to use a loop diuretic.
- Monitor patients for electrolyte and volume depletion by following their potassium, magnesium, BUN, and creatinine levels. This is especially true for those on combination therapy.
- Excessive diuresis may result in:

    1. Prerenal azotemia
    2. Orthostatic hypotension
    3. Hypokalemia and hypomagnesemia
    4. Inability to achieve optimal dose of ACE inhibitor

- Hyponatremia is an indication for fluid restriction in a volume overloaded patient and a decrease in diuretic in a volume depleted patient.
- Hypokalemia indicates that the patient has been diuresed without adequate potassium supplementation. If hypokalemia is a chronic problem, a potassium-sparing diuretic should be considered.
- Hyperkalemia may be the result of too much potassium supplementation, potassium-sparing diuretics, digoxin toxicity, ACE inhibitor intolerance, or renal insufficiency.
- Hypomagnesemia often accompanies hypokalemia. If high doses of diuretic are used, serum magnesium levels should be checked regularly and oral supplementation given as indicated. Hypomagnesemia may prevent correction of hypokalemia.
- Orthostatic hypotension may indicate overdiuresis in the absence of congestive symptoms and may be accompanied by an increased BUN to creatinine ratio. If volume depletion is not present, intolerance of

the ACE inhibitor is likely. (See "Hypotension," under "ACE Inhibitors.")
- See Annotation Appendix E, "Oral Diuretic Dosages" in the original guideline document for information on dosing diuretics.

Evidence supporting this recommendation is of classes: A, C, D

Spironolactone

- A multi-center, randomized clinical trial showed a reduction in mortality among patients with Class III-IV heart failure who were treated with spironolactone 12.5 to 25 mg per day. These patients were already on stable doses of digoxin and ACE inhibitors.

Evidence supporting this recommendation is of class: A

Inotropes

Currently, none of the inotropes available for the treatment of severe decompensated heart failure have demonstrated the ability to improve mortality. A review of the literature has in fact shown an increase in mortality with the use of these agents. [Conclusion Grade I: See Conclusion Grading Worksheet – Appendix B – Annotation #5 (Inotropes) in the original guideline document] The use of inotropes should therefore be restricted to those patients needing symptomatic relief who are no longer responding to other therapies. [Conclusion Grade III: See Conclusion Grading Worksheet – Appendix B – Annotation #5 (Inotropes) in the original guideline document]

Evidence supporting this recommendation is of classes: A, D, M

Digoxin:

- Digoxin should be used in patients with left-ventricular systolic dysfunction if there is symptomatic evidence of elevated filling pressures, a third heart sound, ventricular dilation, or very depressed ejection fraction.
- Digoxin is a useful drug in heart failure patients with atrial fibrillation with a rapid ventricular response.
- Digoxin should be added in symptomatic patients who are already managed with ACE inhibitors and diuretics.
- The initiation of digoxin in asymptomatic heart failure patients still remains controversial.
- Loading doses are generally not needed and steady state generally takes one week to reach (longer in patients with renal impairment).
- Serum levels of 0.7 to 1.5 ng/ml are considered therapeutic although levels up to 2.5 ng/ml may be tolerated. Serum levels do not always correlate to symptoms of digoxin toxicity.
- Monitor symptoms of toxicity (nausea, confusion, visual disturbance, anorexia), reduction of renal function, or conduction abnormality.
- To avoid digitalis toxicity, care should be used to:

1. Use lower doses in the elderly and those with renal impairment.
2. Check digitalis level in one to two weeks after start of therapy in elderly or renal impaired patients.
3. Beware of drug interactions with new medications. See Annotation Appendix G, "Potential Drug- Drug Interactions" in the original guideline document.

Digitalis improves symptoms, exercise tolerance, and quality of life, but neither increases nor decreases mortality. Refer to the ICSI guideline Heart Failure in Adults Annotations #6 and 14 (Digitalis).

Evidence supporting this recommendation is of classes: A, D

Dobutamine (intravenous):

- Dobutamine is FDA approved for use in heart failure patients with stable blood pressure (systolic blood pressure [SBP] greater than 100 mm Hg) when parenteral therapy is necessary for inotropic support in the short-term treatment of adults with cardiac decompensation due to depressed contractility resulting either from organic heart disease or from cardiac surgical procedures.
- A meta-analysis of inotropes in heart failure found a trend to excess mortality with inotropic therapy, with no evidence that beta agonists and phosphodiesterase (PDE) inhibitors were different in this regard.
- While studies have shown symptomatic improvement in patients on continuous dobutamine infusion at 5 to 7.5 micrograms/kg/min for up to 5 days, this group has often found lower doses of 2 to 5 micrograms/kg/min to be sufficient.
- The use of intermittent and continuous intravenous dobutamine infusions have produced conflicting results. Due to the potential risk for increased morbidity and mortality, this therapy should be reserved for those patients with severe decompensated heart failure who are no longer responding to other therapies.
- Concomitant beta blockers can reduce the inotropic response to beta agonists, which may reduce both any benefit or harm from these agents. The clinical significance of this is unclear.
- There are no placebo-controlled data documenting improved survival from either intermittent or continuous intravenous dobutamine.
- Contraindications to the use of dobutamine include hypersensitivity to dobutamine and the presence of idiopathic hypertrophic subaortic stenosis.
- Precautions include:

    1. Patients on dobutamine must be monitored for arrhythmias.
    2. Hypovolemia should be corrected with sufficient fluid prior to initiation of dobutamine.
    3. Dobutamine may be ineffective in patients who have recently received a beta blocker; in such cases peripheral vascular resistance may increase.
    4. Patients on dobutamine should have their blood pressure and electrocardiogram (EKG) monitored continuously. In addition,

wedge pressure and cardiac output should also be followed whenever possible.
5. Patients on dobutamine should have their serum potassium levels drawn regularly to monitor for hypokalemia.
6. Clinical experience with dobutamine following myocardial infarction has been insufficient to establish the safety of drug for this use. There is concern in the literature that using dobutamine during an acute ischemic event may actually increase the size of the infarction.
7. No improvement may be seen in the presence of marked mechanical obstructions (e.g., severe aortic stenosis).

Evidence supporting this recommendation is of classes: A, D, M

Dopamine (intravenous):

- Dopamine is FDA approved for use in hypotensive heart failure patients (systolic blood pressure less than 100 mm Hg) when parenteral therapy is necessary for inotropic support in the short-term treatment of adults with cardiac decompensation.
- A meta-analysis of inotropes in heart failure found a trend to excess mortality with inotropic therapy with no evidence that beta-agonists and PDE inhibitors were different in this regard.
- Dopamine is effective in achieving desirable hemodynamics in some patients with refractive heart failure associated with acute oliguric renal failure.
- Outpatient dopamine infusions may be an effective form of therapy for selected patients with severe heart failure who are refractory to more conventional treatment or who are awaiting cardiac transplantation.
- Low (renal) dose dopamine has no beneficial effects on renal function in heart failure.
- There are no placebo-controlled data documenting improved survival from either intermittent or continuous intravenous dopamine.
- Contraindications to dopamine use include:

    1. Hypersensitivity to dopamine
    2. Use in patients with pheochromocytoma
    3. Use in patients with uncorrected tachyarrhythmias or ventricular fibrillation
- Precautions with dopamine use include:

    1. During dopamine infusion the following parameters should be monitored: urine output, cardiac output, blood pressure, and (if feasible) pulmonary wedge pressure.
    2. Prior to treatment with dopamine, hypovolemia should be fully corrected.
    3. Hypoxia, hypercapnia, and acidosis must be identified and corrected prior to using dopamine.
    4. If an increased number of ectopic beats are observed, the dose should be reduced if possible.
    5. If a disproportionate rise in the diastolic pressure is observed in patients receiving dopamine, the infusion rate should be

decreased and the patient observed carefully for further evidence of predominant vasoconstrictor activity.

6. At lower infusion rates, if hypotension occurs, the infusion rate should be rapidly increased until adequate blood pressure is obtained. If hypotension persists, dopamine should be discontinued and a more potent vasoconstrictor agent such as norepinephrine should be administered.
7. Dopamine should be infused into a large vein whenever possible to prevent the possibility of infiltration of perivascular tissue adjacent to the infusion site.
8. Patients with a history of occlusive vascular disease (e.g., arteriosclerosis, arterial embolism, Raynaud's disease, cold injury such as frostbite, diabetic endarteritis, and Buerger's disease) should be closely monitored for any changes in color or temperature of the skin of the extremities.

Evidence supporting this recommendation is of classes: D, M

Milrinone (intravenous):

- Intravenous milrinone is FDA approved for use in heart failure patients with stable blood pressure (systolic blood pressure >100 mm Hg) when parenteral therapy is necessary for inotropic support in the short-term treatment of adults with cardiac decompensation.
- A meta-analysis of inotropes in heart failure found a trend to excess mortality with inotropic therapy with no evidence that beta-agonists and PDE inhibitors were different in this regard.
- Short- and long-term intravenous milrinone can provide hemodynamic and symptomatic relief in patients with advanced heart failure.
- Milrinone may have a bi-directional effect based on etiology in decompensated heart failure. Milrinone may be deleterious in ischemic heart failure, but neutral to beneficial in non-ischemic cardiomyopathy.
- The majority of experience with intravenous milrinone has been in patients receiving digoxin and diuretics.
- There is no experience in controlled trials with infusions of milrinone for periods exceeding 48 to 72 hours.
- Currently, oral phosphodiesterase inhibitors have no place in heart failure treatment.
- Milrinone is contraindicated in patients who are hypersensitive to it.
- Precautions with milrinone use include:

    1. Milrinone should not be used in patients with severe obstructive aortic or pulmonic valvular disease in lieu of surgical relief of the obstruction. Like other inotropic agents, it may aggravate outflow tract obstruction in hypertrophic subaortic stenosis.
    2. Supraventricular and ventricular arrhythmias have been observed in the high-risk population treated. In some patients milrinone has been shown to increase ventricular ectopy, including nonsustained ventricular tachycardia. Therefore, patients receiving milrinone should be closely monitored during infusion.

3. Milrinone produces a slight shortening of atrioventricular (AV) node conduction time, indicating a potential for an increased ventricular response rate in patients with atrial flutter/fibrillation which is not controlled with digitalis therapy.
4. During therapy with milrinone, blood pressure and heart rate should be monitored and the rate of infusion slowed or stopped in patients showing excessive decreases in blood pressure.
5. If prior vigorous diuretic therapy is suspected to have caused significant decreases in cardiac filling pressure, milrinone should be cautiously administered.
6. No clinical studies have been conducted in patients in the acute phase of post myocardial infarction. Until further clinical experience with this class of drugs is gained, milrinone is not recommended in these patients.
7. Fluid and electrolyte changes and renal function should be carefully monitored during therapy with milrinone. Improvement in cardiac output with resultant diuresis may necessitate a reduction in the dose of diuretic. Potassium loss due to excessive diuresis may predispose digitalized patients to arrhythmias. Therefore, hypokalemia should be corrected by potassium supplementation in advance of or during use of milrinone.
8. Reductions in infusion rate may be necessary in patients with renal impairment.

Evidence supporting this recommendation is of classes: A, C, M

Beta Blockers

- Titrate to heart rate greater than 60, hold if heart rate less than 60.
- Studies strongly support use of beta blockers that have demonstrated reductions in mortality (e.g., carvedilol, metoprolol succinate, bisoprolol) in patients with Class I-IV congestive heart failure (CHF). Recent data from COMET demonstrated carvedilol to have a 17% risk reduction in mortality over metoprolol tartrate.
- Beta blockers having demonstrated reductions in mortality should be considered in patients who develop congestive heart failure following acute myocardial infarction, and who can tolerate the negative ionotropic effects. The beta blocker should be started as soon as the patient is hemodynamically stable.
- Beta blockers should be started at the smallest possible dose. Carvedilol 3.125 mg twice a day (BID) and titrate as tolerated up to 25 mg BID maximum (50 mg BID for patients with mild to moderate heart failure >85 kg), metoprolol succinate 12.5 once daily for NYHA Classes III-IV or 25 mg once daily for Class II, to increase the dose every 2 weeks to the maximum tolerated dose. Cutting the tablet to achieve accurate initial dosing may be difficult for some patients (i.e., one-quarter of a 25 mg metoprolol succinate tablet).
- When the patient is hemodynamically stable, beta blockers should be started in the inpatient setting. Beta blockers decrease hospitalizations and mortality. Stable is defined as:
    - Not hospitalized in an intensive care unit

    - No or minimal evidence of fluid overload or volume depletion
    - Did not require recent treatment with an intravenous positive inotropic agent
- See Annotation Appendix F, "Comparison of Commonly Used Beta Blockers" in the original guideline document. After appropriate stabilization, it is safe to start beta blockers in the inpatient setting. Beta blockers decrease hospitalizations and mortality. [Conclusion Grade I: See Conclusion Grading Worksheet – Appendix C – Annotation #5 (Beta blockers) in the original guideline document]
- Precautions

  Beta blockers should be used with caution in those patients with reactive airways disease and avoided in those patients with symptomatic bradycardia or advanced heart block (unless treated by pacemaker).

- Brand carvedilol and brand metoprolol succinate are both more expensive than generic beta blockers although these are the only two drugs currently approved for heart failure and that have demonstrated mortality benefits.

Evidence supporting this recommendation is of classes: A, M

Antiarrhythmics

- Antiarrhythmics are not indicated for the suppression of ventricular premature beats or non-sustained ventricular tachycardia, which are either asymptomatic or perceived as palpitations.
- In patients with atrial fibrillation, the decision to use or not to use an antiarrhythmic to maintain sinus rhythm may depend on how well-tolerated the atrial fibrillation is from a hemodynamic standpoint.

The preponderance of data suggests that antiarrhythmics, when used empirically for ventricular tachycardia or for the suppression of atrial fibrillation, increase mortality. Amiodarone is an exception to this rule and is probably mortality neutral.

Refer to the ICSI guideline Heart Failure in Adults Annotations #6 and 14 (Antiarrhythmics).

Evidence supporting this recommendation is of classes: A, C, X

Anticoagulants

- Anticoagulation with warfarin is indicated in heart failure patients with atrial fibrillation mechanical heart valves or in patients with impaired systolic function (i.e., EF less than 20%) prior thromboemboli and left ventricular mural thrombi.

Refer to the National Guideline Clearinghouse (NGC) summary of the ICSI guideline Anticoagulant Therapy Supplement

Evidence supporting this recommendation is of classes: A, B

6. Non-Invasive and Invasive Imaging/Physiologic Assessment

Diagnostic Tests

- Electrocardiogram
- Chest radiograph
- Assessment of ventricular function (echocardiogram, radionuclide ventriculography) - Use clinical judgment to reassess ventricular function if an evaluation has been done within the last 6 months.

Specific etiologies of ventricular dysfunction:

Diastolic dysfunction of mild degree is commonly associated with systolic dysfunction, but isolated diastolic dysfunction may be seen with left ventricular hypertrophy, myocardial ischemia, constrictive pericarditis or pericardial tamponade, or in the case of infiltrative diseases such as amyloidosis.

Evidence supporting this recommendation is of classes: C, R

Interpretation of Ventricular Function Testing

Heart failure is a clinical syndrome that correlates poorly with ejection fraction. Some patients may have a combination of systolic and diastolic dysfunction. Measurement of left ventricular (LV) function provides important prognostic information.

Objective assessment of left ventricular function is necessary because chest x-ray, ECG, and history and physical often fail to distinguish normal from low ejection fraction in patients with heart failure. See ICSI Heart Failure guideline Annotation #3.

Measurement may change with changes in the underlying disease process or with differences in systolic or diastolic ventricular loading conditions. Hence, they may change over time because of progression or regression of the underlying ventricular muscle dysfunction, and/or with changes in therapy. It is reasonable to reassess ventricular function after interventions or when symptoms have changed significantly. Changes in ventricular function may imply a change in prognosis and may require changes in therapy.

Evidence supporting this recommendation is of class: C

Refer to the original guideline document for information on advantages and disadvantages of diagnostic tests (echocardiogram, radionuclide ventriculogram, left ventriculogram, magnetic resonance imaging [MRI], and right heart catheterization [Swan-Ganz]).

The quantitative assessment of systolic or diastolic dysfunction does not imply an understanding of the underlying etiology of the ventricular dysfunction.

Care must be taken to determine the cause of dysfunction so that specific therapy can be instituted (e.g., treatment of ischemia, valve disease, hypertension, pericardial disease, hyperthyroidism).

7. Progressive Deterioration?

Referral to specialist

- Class III and IV symptoms refractory to medical management
- Rapidly progressive symptoms in spite of maximal medical management
- Patients with syncope of unknown cause or those who have undergone cardioversion for ventricular tachycardia or fibrillation
- Patients in whom moderate doses of vasodilating drugs cannot be tolerated
- Young people with either severe left ventricular dysfunction, severe left ventricular dilation, or significant valvular regurgitation. Many of these patients may be candidates for cardiac transplantation or other cardiac surgical procedures.
- Early referral should be considered even in patients with minimal symptoms.
- Consider referral for NYHA Class III patients for biventricular pacing who are already on optimized medical therapy.

9. Non-Pharmacologic Management

Patient education should be initiated at this time.

Diet and Alcohol Intake

Dietary indiscretion remains a common cause of exacerbation of heart failure, and reinforcement of the importance of dietary adherence should occur at each interaction.

1. Patients should be advised to avoid excessive fluid intake, but not all patients require a fluid restriction. If patient is edematous, a 2,000 cc/day fluid restriction should be recommended.
2. A reduction in dietary sodium intake of 2,000 to 3,000 mg per day alone may provide substantial hemodynamic and clinical benefits for heart failure patients. Unfortunately patients (and physicians) frequently rely solely on diuretics to control symptoms. Stress the importance of reading labels and a no-salt-added diet. If patient has repeat episodes of edema or failure, a more strict recommendation is appropriate. Example of a stricter recommendation: 2,000 mg Na^+/day, and not more than 700 mg/meal.
3. Caution patients about the use of potassium-containing salt substitutes which could contribute to the development of significant hyperkalemia.
4. Assess usual diet, checking for commonly used foods, ethnic foods, or special diets and practices. Avoid overly restrictive diet regimens unless medically necessary.
5. Alcohol use should be discouraged, at the least saved for special occasions. One drink is considered 10 oz. of beer, 5 oz. of wine, or 1.5

oz. of hard liquor. In severe heart failure, complete abstinence is recommended.

6. Handouts and educational guides, while helpful, may be inadequate for many patients, and a dietary consultation is recommended. The goal of dietary instruction and consultation is also to help patients prioritize various dietary restrictions and to develop meal plans based on these recommendations.

Daily Weights

Patients should weigh themselves daily on the same scale, wearing the same amount of clothing. Patients keep an ongoing record of these weights. Daily weights should be taken upon rising in the morning (before eating and after urinating). Patients should call their health care provider if a 2 pound weight gain or more overnight or a 5 pound weight gain or more in a week. To avoid dehydration, patients should additionally call their health care provider if they have decreased oral intake of fluids and are experiencing unanticipated weight loss of greater than 3 to 5 pounds.

Exercise

1. Patients should be advised that if they are overly tired the day following an exercise session, modifications are in order. Patients should incorporate an appropriate warm-up and cool-down period. Referral to a cardiac rehabilitation program for exercise prescription and modeling will contribute to patients' compliance with exercise, functional improvement, and quality of life. Participation in a formal program may also contain education and compliance monitoring of lifestyle management components for heart failure.
2. Patients should be counseled about the benefits of a low-intensity aerobic exercise and light weight conditioning programs. Abnormal responses to exercise, such as lightheadedness, chest pain, marked dyspnea, or unusual fatigue, should also be discussed with the patient. Increased workload on the heart, either too heavy or too sustained, may result in decompensation of heart failure. Modifications to a patient's daily work schedule and duties may becomes necessary to accommodate the need for more frequent rest breaks and decreased functional abilities. See general guidelines for exercise training of patients with heart failure, as stated by Sullivan and Hawthorne (Sullivan MJ, Hawthorne MH. Nonpharmacologic interventions in the treatment of heart failure. J Cardiovas Nurs 10:47-57, 1996). See ICSI Heart Failure in Adults guideline as well.

   Note: It is not uncommon for patients who have been exercising for approximately 6 weeks to need an increase in diuretic dosage. Care should be taken that this does not discourage the patient from continuing exercise training.

Sleep

The hospitalized heart failure patient should be observed during sleep by nursing personnel for evidence of sleep apnea and nocturnal oxygen

desaturations. The incidence of central and obstructive sleep apneas in this population is significant, and appropriate assessment for the presence of this condition and treatment if observed is essential for improving the patient's prognosis. The incidence of sudden cardiac death is increased in heart failure patients with untreated sleep apnea. Referral for sleep study should be considered when signs or symptoms of sleep apnea are detected.

Stress Reduction

Encourage relaxation response training to decrease the workload on the heart.

Evidence supporting this recommendation is of classes: A, C, M, R

10. Discharge Plan with Specific Therapeutic Goals

Prevention of symptom exacerbation

If available, consider referral to a heart failure clinic or case manager if the patient has medical problems or is at high risk for re-hospitalization

Accessibility

1. To prevent heart failure exacerbation, efforts and resources should be directed toward early intervention in the form of increased accessibility to care and education aimed at symptom recognition and treatment plan adherence.
2. Frequently, patients wait until they are in crisis before seeking medical assistance, bypassing the physician's office and going straight to the Emergency Department (ED). Limited hours and limited/untrained staff at physicians' offices have been cited as reason patients seek acute care with worsening symptoms of heart failure.
3. Case managers and Heart Failure clinics may be effective strategies to avert ED visits and hospitalizations by providing patients with a contact person who is familiar with their care to expedite treatment alternatives. This contact person, usually a nurse, is available to answer questions and clarify instructions, potentially increasing treatment plan compliance. The nurse should coordinate adequate ancillary support services available (i.e., social workers, dietary, etc.).
4. Time between visits is important for the patient to formulate questions and assimilate the previously presented information. Family members and care givers should also be involved in education to support the patient's efforts.

Medications

1. Because of the advanced age of this population and the complexity of medication regimes, every effort should be made to simplify and clarify a patient's medications.
    - Group medications so they are taken together (i.e., not more than 4 times per day).

- Cut down on the frequency of each medication taken per day (i.e., twice daily [BID] versus three times a day [TID] if bioequivalent).
- Emphasize taking medications at the appropriate time to maximize symptom control (i.e., taking nitrates on an empty stomach. However, this may increase the risk of syncope with elderly patients.).
- Patients should be encouraged to avoid non-steroidal anti-inflammatory drugs (NSAIDs) and cyclooxygenase-2 (COX-2) inhibitors.

2. All medication instructions, including over-the-counter medications, should be reviewed at each interaction, written clearly, and reinforced verbally. The indications and possible side effects of each medication should be explained, and patients should be reminded not to stop or change their medications without talking to their physician or nurse.

   See Annotation Appendix H, "Medications That May Worsen/Exacerbate Heart Failure" in the original guideline document.

Evidence supporting this recommendation is of classes: A, R

Emergent Management Algorithm Annotations

12. Initial Patient Assessment
    - History
    - Physical

Notes:

- Patients with decompensated aortic stenosis should not receive vasodilator agents (vs. mitral regurgitation patients that benefit greatly).
- Patients with jugular venous distension from right ventricular infarct may require a fluid challenge.
- Some patients with acute pulmonary edema may have intravascular volume depletion and need a fluid challenge.
- Patients with low cardiac output and peripheral vasoconstriction have unreliable noninvasive blood pressure measures.
- Digoxin, as an inotrope, is not useful in the acute management of decompensated heart failure (may be used to control atrial fibrillation).

Differential diagnosis:

- Chronic obstructive lung disease
- Pulmonary embolism
- Asthma exacerbation
- Sepsis
- Pulmonary embolus
- Volume overload (iatrogenic)
- Severe pneumonia
- Chordae rupture
- Anaphylaxis

- Acute coronary syndrome

15. Initiate O_2 Therapy, Start Intravenous, Order Labs, Chest X-Ray and ECG. Consider Echocardiogram (ECHO)
    A. Initial Laboratory Assessment:
        - Complete blood count (CBC)
        - Electrolytes (Na^+, K^+)
        - Renal function (BUN, creatinine)
        - Magnesium (if on diuretics)
        - Calcium
        - Urinalysis
        - Digoxin level (if on digoxin)
        - Prothrombin time (PT)/international normalized ratio (INR) if on Coumadin
        - Cardiac markers (creatine kinase [CK], troponin)
        - Glucose
        - Brain natriuretic peptide (BNP) (if the diagnosis is uncertain)
        - Blood gases (may be indicated if the patient is hypoxic, has underlying lung disease, or has persistent respiratory distress).
    B. ECG and continuous rhythm monitoring: Recommended in all cases.
    C. Imaging: A chest x-ray is recommended in all cases.

    An emergent echocardiogram is indicated for the patient who is not improving with initial interventions.

22. Assess Blood Pressure, Perfusion, and Volume Status

    Refer to original guideline document for information on bedside hemodynamic assessment.

27. Acute Pulmonary Edema BP >100 mm Hg

    Patients with edema, jugular venous distension, positive hepatojugular reflux, and/or rales are likely to be volume overloaded. See Annotation Appendix A, "Emergent Heart Failure Management" in the original guideline document.

    Causes of Acute Pulmonary Edema:

    - Acute myocardial infarction/ischemic heart disease
    - Acute Renal failure
    - Pericardial tamponade
    - Hyper/hypothyroidism
    - Obstructive or severe valvular disease
    - Arrhythmias
    - Cardiomyopathy (e.g., infectious, toxic, hypothyroidism, peripartum, hypertrophic)
    - Malignant hypertension
    - Pulmonary embolus
    - Hypermetabolic conditions (thyrotoxicosis, pheochromocytoma, heat stroke)

29. Loop Diuretics: Intravenous Bolus, Consider Intravenous Infusion

Refer to the original guideline document for details on loop diuretics dosing, routes of administration, and pharmacologic characteristics.

30. Nitroglycerin Sublingual or Paste

Concurrent with diuretic therapy is the initiation of vasodilators

- Nitroglycerine, 0.4 mg sublingual or paste

Many patients will improve symptomatically with the "first-line therapy" and may be transferred to an observation unit or inpatient bed.

Evidence supporting this recommendation is of class: R

32. Nitroglycerine Infusion

Patients who are not improving will need more aggressive treatment.

- Begin nitroglycerine infusion at 10 to 20 micrograms/min and increase by 10 to 20 micrograms/min every 3 to 5 minutes to achieve desired effect. The maximum dose is 300 micrograms/min.
- The guideline developers recommend the upward titration of nitroglycerine before converting to nesiritide.

Alternative dosing protocols exist which may provide a greater safety margin, such as: intravenous: non-polyvinylchloride (PVC) tubing, 5 micrograms/min, initial titration should be in 5 micrograms/min increments at intervals of 3 to 5 min guided by patient response; if no response is seen at 20 micrograms/min, incremental increases of 10 and 20 micrograms/min may be used; PVC tubing, initial dose 25 micrograms/min intravenously.

Additional note: some intensive care units (ICUs) and EDs are now titrating in micrograms/kg/min.

Evidence supporting this recommendation is of class: R

37. Change to Nesiritide

Patients who continue to exhibit signs and symptoms of volume overload despite aggressive loop diuretics and intravenous nitroglycerine may be a candidate for Nesiritide.

- Nesiritide 2 micrograms intravenous bolus, then 0.01 microgram/kg/min intravenous infusion
- Nesiritide, a natriuretic peptide (see Annotation #7, "Progressive Deterioration?"), has been tested in combination with diuretics but not

with intravenous nitroglycerine. The safety profile is favorable in comparison with the phosphodiesterase inhibitors (e.g., milrinone) or the adrenergic inotropes (e.g., dobutamine).

The experience with nesiritide to date has been limited, in comparison with the other two drugs.

Evidence supporting this recommendation is of class: A

43. Stabilized?

Patients who stabilize may be admitted to an observation unit or monitored hospital bed.

Unstable criteria include:

- Unstable vital signs – blood pressure (BP) 220/120 mm Hg, respiration rate (RR) greater than 25, heart rate (HR) greater than 130 beat per minute (bpm)
- ECG or serum markers of myocardial ischemia greater than 90%
- Complex decompensation (concomitant end-organ hypoperfusion, volume overload, and systemic vasoconstriction)
- Requiring continuous vasoactive medication (e.g., nitroglycerin, nitroprusside, dobutamine, or milrinone) to stabilize hemodynamics
- Nonsustained ventricular tachycardia not caused by electrolyte imbalance
- Acute mental status abnormality
- Severe electrolyte imbalances

46. ICU Admission/Consider Consultation and/or Tertiary Center Referral

Patients that remain unstable are candidates for ICU admission or tertiary care center referral. Consider consultation for emergent imaging and physiologic assessment, either non-invasive or invasive (e.g., pulmonary artery catheterization). This evaluation will allow more precise medical management, and will determine candidates for mechanical devices, continuous inotropic therapy, biopsy, surgical interventions, or hospice.

48. ED observation or Short Stay Candidate?

Some heart failure patients may be managed in a short stay or observation unit. A short stay for diagnosis, intensive therapy, and education has demonstrated advantages. Institutions which utilize observation units will need to have selection criteria and observation protocols to achieve optimal results.

Evidence supporting this recommendation is of class: R

Definitions:

Classes of Research Reports:

A. Primary Reports of New Data Collection:

Class A:

- Randomized, controlled trial

Class B:

- Cohort study

Class C:

- Non-randomized trial with concurrent or historical controls
- Case-control study
- Study of sensitivity and specificity of a diagnostic test
- Population-based descriptive study

Class D:

- Cross-sectional study
- Case series
- Case report

B. Reports that Synthesize or Reflect upon Collections of Primary Reports

Class M:

- Meta-analysis
- Systematic review
- Decision analysis
- Cost-effectiveness study

Class R:

- Consensus statement
- Consensus report
- Narrative review

Class X:

- Medical opinion

Conclusion Grades:

Grade I: The evidence consists of results from studies of strong design for answering the question addressed. The results are both clinically important and consistent with minor exceptions at most. The results are free of significant doubts about generalizability, bias, and flaws in research design. Studies with negative results have sufficiently large samples to have adequate statistical power.

Grade II: The evidence consists of results from studies of strong design for answering the question addressed, but there is uncertainty attached to the conclusion because of inconsistencies among the results from different studies or because of minor doubts about generalizability, bias, research design flaws, or adequacy of sample size. Alternatively, the evidence consists solely of results from weaker designs for the question addressed, but the results have been confirmed in separate studies and are consistent with minor exceptions at most.

Grade III: The evidence consists of results from studies of strong design for answering the question addressed, but there is substantial uncertainty attached to the conclusions because of inconsistencies among the results from different studies or because of serious doubts about generalizability, bias, research design flaws, or inadequacy of sample sizes. Alternatively, the evidence consists solely of results from a limited number of studies of weak design for answering the question addressed.

Grade Not Assignable: There is no evidence available that directly supports or refutes the conclusion.

CLINICAL ALGORITHM(S)

A detailed and annotated clinical algorithm is provided for:

- Inpatient Management
- Emergent Management
- Acute Pulmonary Edema

EVIDENCE SUPPORTING THE RECOMMENDATIONS

TYPE OF EVIDENCE SUPPORTING THE RECOMMENDATIONS

The guideline contains an annotated bibliography and discussion of the evidence supporting each recommendation. The type of supporting evidence is classified for selected recommendations (see "Major Recommendations").

In addition, key conclusions contained in the Work Group's algorithm are supported by a grading worksheet that summarizes the important studies pertaining to the conclusion. The type and quality of the evidence supporting these key recommendations (i.e., choice among alternative therapeutic approaches) is graded for each study.

BENEFITS/HARMS OF IMPLEMENTING THE GUIDELINE RECOMMENDATIONS

POTENTIAL BENEFITS

Overall Benefits

Appropriate medical management of patients with heart failure

Specific Benefits

- Angiotensin-converting enzyme (ACE) inhibitors slow disease progression, improve exercise capacity, and decrease hospitalization and mortality.
- Angiotensin receptor blockers (ARBs) reduce afterload, improve cardiac output, and are easy to use, which could potentially increase patient compliance. There are conflicting data when adding an ARB to ACE inhibitor or to ACE inhibitor and a beta blocker. Some trials show benefit while others do not.
- Thiazide and loop diuretics are equally effective in mild heart failure while loop diuretics are more effective in severe heart failure. Combined diuretic therapy has been shown to be useful in refractory cases of volume overload
- A multi-center, randomized clinical trial showed a reduction in mortality among patients with Class III-IV heart failure who were treated with spironolactone 12.5 to 25 mg/day. These patients were already on stable doses of digoxin and ACE inhibitors.
- Hydralazine combined with isosorbide dinitrate has been shown to reduce mortality and increase exercise tolerance in patients with symptomatic heart failure.
- Inotropes can provide symptomatic relief. Digoxin improves symptoms for patients in sinus rhythm with ventricular dilatation, elevated filling pressures, and a third heart sound. Digitalis improves symptoms, exercise tolerance, and quality of life, but neither increases nor decreases mortality. Dopamine is effective in achieving desirable hemodynamics in some patients with refractive heart failure associated with acute oliguric renal failure. Milrinone can provide hemodynamic and symptomatic relief in patients with advanced heart failure.
- Beta blockers decrease hospitalization, mortality, and reinfarction.
- A reduction in dietary sodium intake of 2,000 to 3,000 mg/day alone may provide substantial hemodynamic and clinical benefit.
- Nitrates have a beneficial effect on hemodynamics in heart failure, but the data on mortality effects are sparse.
- Refer to the original guideline document for information on the advantages of diagnostic tests.

POTENTIAL HARMS

Adverse Effects of Medications

- Angiotensin-converting enzyme (ACE) inhibitors may cause hypotension, hyperkalemia, renal insufficiency, angioedema, and intractable cough.
- Angiotensin receptor blockers are as likely as ACE inhibitors to produce hypotension, worsening renal function, and hyperkalemia.
- Refer to "Major Recommendations" field and Annotation Appendix A, "Emergent Heart Failure Management" in the original guideline document for information on precautions with and common side effects of nitroglycerin, nitroprusside, and nesiritide (Natrecor®).
- Thiazide and loop diuretics may cause postural hypotension, hypokalemia, hypomagnesemia, hyperglycemia, hyperuricemia, rash rarely, pancreatitis, bone marrow suppression, and anaphylaxis.
- Spironolactone may cause gynecomastia.
- Inotropes may cause arrhythmias. A meta-analysis of inotropes in heart failure found a trend toward excess mortality with inotropic therapy with no evidence that beta-agonists and PDE inhibitors were different in this regard.

In addition, dobutamine and milrinone can produce tachycardia; dopamine and digoxin may cause nausea and vomiting. Other adverse events of inotropes include: Dobutamine: the use of intermittent and continuous intravenous dobutamine infusions may be associated with little efficacy and may increase the risk of death; Milrinone: hypotension; Dopamine: volume depletion; Digoxin: visual disturbance, confusion, anorexia, reduction of renal function, and conduction abnormality.

- Beta blockers should be used with caution in patients with reactive airways disease and avoided in patients with symptomatic bradycardia or advanced heart block (unless treated by pacemaker).
- The preponderance of data suggests that antiarrhythmics, when used empirically for ventricular tachycardia or for the suppression of atrial fibrillation increase mortality. Amiodarone is an exception to this rule and is probably mortality neutral.

Adverse Effects of Non-Pharmacologic Management

Abnormal response to exercise, such as lightheadedness, chest pain, marked dyspnea, or unusual fatigue requires modifications in exercise program.

Disadvantages of Diagnostic Tests

Refer to the original guideline for information on disadvantages of diagnostic tests.

Potential Drug-Drug Interactions

Refer to Annotation Appendix G "Potential drug-drug interactions" in the original guideline for details on potential drug interactions with ACE inhibitors, digoxin, diuretics, carvedilol, metoprolol, and bisoprolol.

CONTRAINDICATIONS

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Contraindications

- Angiotensin-converting enzyme (ACE) inhibitors and angiotensin receptor blockers are contraindicated in patients with history of adverse reactions to these agents, serum potassium greater than 5.5 meq/L, symptomatic hypotension (unless due to excessive diuresis), severe renal artery stenosis, and pregnancy. ACE inhibitors are also contraindicated in patients with cough, rash side effects, and known hypersensitivity to ACE inhibitors.
- Nitroglycerin is contraindicated in patients with a known hypersensitivity or idiosyncrasy reaction to nitroglycerin and other organic nitrates, or nitrites; hypotension or uncorrected hypovolemia, as the use of nitroglycerin in such states could produce severe hypotension or shock; increased intracranial pressure (e.g., head trauma or cerebral hemorrhage); inadequate cerebral circulation; and constrictive pericarditis and pericardial tamponade.
- Contraindications to nitroprusside include using nitroprusside in the treatment of compensatory hypertension, where the primary hemodynamic lesion is

aortic coarctation or arteriovenous shunting; using nitroprusside to produce hypotension during surgery in patients with known inadequate cerebral circulation or in moribund patients (A.S.A. Class 5E) coming to emergency surgery. Nitroprusside should be avoided in patients with congenital (Leber's) optic atrophy or with tobacco amblyopia (a rare form of amblyopia combining nutritional deficiency and tobacco sensitivity).

- Contraindications to nesiritide include hypersensitivity to nesiritide or any of its components, hypotension with a systolic blood pressure less than 90 mm Hg, and using nesiritide in patients with cardiogenic shock.
- Contraindications to dobutamine include hypersensitivity to dobutamine and the presence of idiopathic hypertrophic subaortic stenosis.
- Dopamine is contraindicated in patients with hypersensitivity to dopamine, pheochromocytoma, and uncorrected tachyarrhythmias or ventricular fibrillation.
- Milrinone is contraindicated in patients who are hypersensitive to it.

QUALIFYING STATEMENTS

QUALIFYING STATEMENTS

- These clinical guidelines are designed to assist clinicians by providing an analytical framework for the evaluation and treatment of patients and are not intended either to replace a clinician's judgment or to establish a protocol for all patients with a particular condition. A guideline will rarely establish the only approach to a problem.
- This clinical guideline should not be construed as medical advice or medical opinion related to any specific facts or circumstances. Patients are urged to consult a health care professional regarding their own situation and any specific medical questions they may have.
- The inpatient guideline for stable heart failure patients has a solid foundation in evidence-based medicine. In contrast, the acute pulmonary edema (APE) guideline surfaces areas of weak or absent evidence for some traditional therapies.
- There are no evidence-based, generally accepted, comprehensive treatment guidelines for decompensated heart failure in the literature. Some of the medications are well researched, others are not. There are no therapeutic trials defining the optimal sequence of drug interventions (e.g., diuretics, inotropes, vasodilators, or neurohormonal) for this condition. Further, there are few "head to head" trials of drugs between drug classes. Some treatments in current practice (e.g., intravenous angiotensin converting enzyme inhibitors, long-term intravenous dobutamine) are not included because the published reports involved very small numbers of patients. Milrinone and dobutamine, although well known to change hemodynamic measures, are not associated with improved clinical outcomes, and may, in fact, increase mortality.
- Morphine is considered to be a first-line drug of choice in many previous recommendations. Again, hemodynamic and anti-anxiety effects are substantiated, but improved clinical outcomes are not. Studies are lacking in the evaluation of potential adverse effects, such as respiratory depression. The negative effects may be overshadowed by the positive effects of other drugs being administered. Interestingly, many recent studies are not including morphine in the first-line therapeutic regimen.

IMPLEMENTATION OF THE GUIDELINE

DESCRIPTION OF IMPLEMENTATION STRATEGY

Once a guideline is approved for release, a member group can choose to concentrate on the implementation of that guideline. When four or more groups choose the same guideline to implement and they wish to collaborate with others, they may form an action group.

In the action group, each medical group sets specific goals they plan to achieve in improving patient care based on the particular guideline(s). Each medical group shares its experiences and supporting measurement results within the action group. This sharing facilitates a collaborative learning environment. Action group learnings are also documented and shared with interested medical groups within the collaborative.

Currently, action groups may focus on one guideline or a set of guidelines such as hypertension, lipid treatment, and tobacco cessation.

The following detailed measurement strategies are presented to help close the gap between clinical practice and the guideline recommendations.

Priority Aims for Medical Groups When Using this Guideline

1. Reduce the mortality and morbidity from heart failure by improving the pharmacologic treatment of patients with heart failure.

   Possible measures for accomplishing this aim:

   a. Percentage of adult patients discharged with a diagnosis of heart failure that are on an angiotensin converting enzyme (ACE) inhibitor at discharge. (Centers for Medicare & Medicaid Services [CMS] Core and Joint Commission on the Accreditation of Health Care Organizations [JCAHO] Core)
   b. Percentage of adult patients discharged with a diagnosis of heart failure (New York Health Association Classes II-IV) that are on a beta blocker with demonstrated reductions in mortality (e.g., carvedilol, metoprolol succinate, bisoprolol) at discharge.
   c. Percentage of adult patients discharged with a diagnosis of heart failure in which the reason for non-use of a beta blocker is documented in the medical record.
   d. Percentage of adult patients discharged with a diagnosis of heart failure in which the reason for non-use of an ACE inhibitor is documented in the medical record.
   e. Mortality rate of patients in a defined heart failure population.
2. Improve the treatment of patients with heart failure by assuring that patients have the etiology and/or precipitating factors of heart failure identified.

   Possible measures for accomplishing this aim:

a. Percentage of adult patients discharged with heart failure that have had an evaluation of left ventricular function before arrival, during hospitalization, or planned after discharge. (CMS Core, JCAHO Core)
b. Percentage of adult patients with heart failure who have a documented evaluation of ischemia, e.g., stress test or angiography, during hospitalization or planned after discharge.
c. Percentage of patients discharged with a diagnosis of heart failure that have a documented etiology in the medical record.

3. Improve care of patients with heart failure by assuring comprehensive follow-up care.

   Possible measures for accomplishing this aim:

   a. Percentage of adult patients admitted with a diagnosis of heart failure that have documentation of discharge counseling (with either a physician or nurse) to include activity level, diet, discharge medications, follow-up appointment, weight monitoring, and what to do if symptoms worsen. (CMS Core, JCAHO Core)
   b. Percentage of adult patients discharged with a diagnosis of heart failure that have a follow-up appointment scheduled within one month after discharge.
   c. Percentage of heart failure patients with a history of smoking cigarettes who are given smoking cessation advice or counseling during hospital stay. (CMS Core and JCAHO Core).

4. Improve care of patients with heart failure by integrating patient feedback.

   Possible measures for this aim:

   a. Percentage of patients who report a positive experience during their admission on a patient satisfaction survey.
   b. Number of heart failure patients who participate in a focus group, advisory group, or improvement team per year.

5. Improve care of patients with heart failure by decreasing the number of hospitalizations of patients with heart failure.

   Possible measures for accomplishing this aim:

   a. Percentage of patients with heart failure who are readmitted for heart failure within 30 days after discharge.
   b. Percentage of patients within a defined heart failure population who present to the Emergency Department (ED) per month.
   c. Percentage of patients within a defined heart failure population that are admitted to the hospital each month for decompensated heart failure.

At this point in development for this guideline, there are no specifications written for possible measures listed above. The Institute of Clinical Systems Improvement (ICSI) will seek input from the medical groups on what measures are of most use as they implement the guideline. In a future version of the guideline, measurement specifications may be included.

INSTITUTE OF MEDICINE (IOM) NATIONAL HEALTHCARE QUALITY REPORT CATEGORIES

IOM CARE NEED

Getting Better
Living with Illness

IOM DOMAIN

Effectiveness
Patient-centeredness
Safety

IDENTIFYING INFORMATION AND AVAILABILITY

BIBLIOGRAPHIC SOURCE(S)

Institute for Clinical Systems Improvement (ICSI). Inpatient management of heart failure. Bloomington (MN): Institute for Clinical Systems Improvement (ICSI); 2004 Feb. 85 p. [169 references]

ADAPTATION

Not applicable: The guideline was not adapted from another source.

DATE RELEASED

2004 Feb

GUIDELINE DEVELOPER(S)

Institute for Clinical Systems Improvement - Private Nonprofit Organization

GUIDELINE DEVELOPER COMMENT

Organizations participating in the Institute for Clinical Systems Improvement (ICSI): Affiliated Community Medical Centers, Allina Medical Clinic, Altru Health System, Aspen Medical Group, Avera Health, CentraCare, Columbia Park Medical Group, Community-University Health Care Center, Dakota Clinic, ENT Specialty Care, Fairview Health Services, Family HealthServices Minnesota, Family Practice Medical Center, Gateway Family Health Clinic, Gillette Children's Specialty Healthcare, Grand Itasca Clinic and Hospital, HealthEast Care System, HealthPartners Central Minnesota Clinics, HealthPartners Medical Group and Clinics, Hutchinson Area Health Care, Hutchinson Medical Center, Lakeview Clinic, Mayo Clinic, Mercy Hospital and Health Care Center, MeritCare, Mille Lacs Health System, Minnesota Gastroenterology, Montevideo Clinic, North Clinic, North Memorial Care System, North Suburban Family Physicians, Northwest Family Physicians, Olmsted Medical Center, Park Nicollet Health Services, Pilot City Health Center, Quello Clinic, Ridgeview Medical Center, River Falls Medical Clinic, Saint

Mary's/Duluth Clinic Health System, St. Paul Heart Clinic, Sioux Valley Hospitals and Health System, Southside Community Health Services, Stillwater Medical Group, SuperiorHealth Medical Group, University of Minnesota Physicians, Winona Clinic, Ltd., Winona Health

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GUIDELINE COMMITTEE

Cardiovascular Steering Committee

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FINANCIAL DISCLOSURES/CONFLICTS OF INTEREST

In the interest of full disclosure, the Institute for Clinical Systems Improvement (ICSI) has adopted the policy of revealing relationships work group members have with companies that sell products or services that are relevant to this guideline topic. The reader should not assume that these financial interests will have an adverse impact on the content of the guideline, but they are noted here to fully inform readers. Readers of the guideline may assume that only work group members listed below have potential conflicts of interest to disclose.

Kristin Ryan, RN, CNP received honoraria from Medtronic and Glaxo Smith Kline Beechman.

Joshua E. Breeding, PharmD received honoraria from Aventis.

No other work group members have potential conflicts of interest to disclose.

ICSI's conflict of interest policy and procedures are available for review on ICSI's website at www.icsi.org.

GUIDELINE STATUS

This is the current release of the guideline.

GUIDELINE AVAILABILITY

Electronic copies: Available from the Institute for Clinical Systems Improvement (ICSI) Web site.

Print copies: Available from ICSI, 8009 34th Avenue South, Suite 1200, Bloomington, MN 55425; telephone, (952) 814-7060; fax, (952) 858-9675; Web site: www.icsi.org; e-mail: icsi.info@icsi.org.

AVAILABILITY OF COMPANION DOCUMENTS

None available

PATIENT RESOURCES

None available

NGC STATUS

This NGC summary was completed by ECRI on June 3, 2004.

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